U.S. Department of Labor

Office of Administrative Law Judges
800 K Street, NW, Suite 400-N
Washington, DC 20001-8002

(202) 693-7300
(202) 693-7365 (FAX)

Issue Date: 04 February 2005

In the Matter of

Martina D. Baird, Widow of
Charles Eugene Baird,
Claimant

v.

Key Mining, Incorporated,
Employer

and

Director, Office of Workers' Compensation Programs,
Party-In-Interest

Case No. 2004BLA5784

DECISION AND ORDER
AWARDING BENEFITS

This proceeding arises from a claim for benefits filed by Martina D. Baird, the surviving spouse of Charles E. Baird, a deceased coal miner, under the Black Lung Benefits Act, 30 U.S.C. §901, et seq. Regulations implementing the Act have been published by the Secretary of Labor in Title 20 of the Code of Federal Regulations.[1]

Black lung benefits are awarded to coal miners who are totally disabled by pneumoconiosis caused by inhalation of harmful dust in the course of coal mine employment and to the surviving dependents of coal miners whose death was caused by pneumoconiosis. Coal workers' pneumoconiosis is commonly known as black lung disease.

A formal hearing was held before the undersigned on October 20, 2004 in Knoxville, Tennessee. At that time, all parties were afforded full opportunity to present evidence and argument as provided in the Act and the regulations. Pursuant to the Briefing Order, dated December 6, 2004, the parties were provided thirty days to submit briefs.

[1] The Secretary of Labor adopted amendments to the "Regulations Implementing the Federal Coal Mine Health and Safety Act of 1969" as set forth in Federal Register/Vol. 65, No. 245 Wednesday, December 20, 2000. The revised Part 718 regulations became effective on January 19, 2001. Since the current claim was filed on April 9, 2001 (DX 3), the new regulations are applicable.

The record consists of the hearing transcript, Director's Exhibits 1 through 23 (DX 1-23), Claimant's Exhibits 1 and 2 (CX 1 and 2), and Employer's Exhibits 1 through 7 (EX 1-7).[2] I have also considered the parties' respective briefs.

The findings of fact and conclusions of law which follow are based upon my analysis of the entire record, including all documentary evidence admitted, arguments made, and the testimony presented.

Procedural History

On May 31, 1994, Mr. Baird filed a claim for benefits as a miner under the Act. Ultimately, he was awarded benefits by Administrative Law Judge Mollie Neal in a decision and order dated April 20, 2000. After appeal by the Employer, the Benefits Review Board remanded the claim, and on December 3, 2002, Judge Neal issued her decision and order awarding benefits.[3] The Employer again appealed, but on February 14, 2003 notified the Board that it wished to withdraw its appeal (DX 1). No additional action was taken on this claim.

On December 11, 2002, Charles Baird passed away; on February 21, 2003, the Claimant, Martina Baird, filed the current application for black lung benefits under the Act, as his surviving spouse (DX 3). On November 17, 2003, the District Director issued a Proposed Decision and Order awarding benefits to the Claimant (DX 18). Following the Employer's timely request for a formal hearing (DX 19), this matter was referred to the Office of Administrative Law Judges for *de novo* adjudication (DX 23), and a hearing was held before the undersigned on October 20, 2004.

Issue

The only issue contested by the Employer is whether Mr. Baird's death was due to pneumoconiosis (Tr. 14).

Applicable Standard

The Regulations at 20 C.F.R. 718 apply to survivors' claims which are filed on or after April 1, 1980. 20 C.F.R. 718.1. Because the Claimant filed her survivor's claim after January 1, 1982, 20 C.F.R. 718.205(c) applies to this claim.

The regulations provide that a survivor is entitled to benefits only where the miner died due to pneumoconiosis. 20 C.F.R. 718.205(a). The Claimant must establish that: (1) the decedent was a coal miner; (2) the decedent suffered from pneumoconiosis at the time of his death; (3) the decedent's pneumoconiosis arose out of his coal mine employment; and (4) the

[2] Also admitted as part of the record for purposes of appeal was Employer's Exhibit 8, a transcript of the deposition of Dr. Caffrey. This exhibit exceeds the evidentiary limitations, and thus has not been considered in making this determination

[3] Judge Neal noted that the Employer stipulated that Mr. Baird had coal workers' pneumoconiosis, and that he was totally disabled.

decedent's death was caused by pneumoconiosis or pneumoconiosis was a substantially contributing cause or factor leading to his death. All elements of entitlement must be established by a preponderance of the evidence. *Strike v. Director, OWCP,* 817 F.2d 395, 399 (7th Cir. 1987). The survivor of a miner who was totally disabled due to pneumoconiosis at the time of death, but died due to an unrelated cause, is not entitled to benefits. 20 C.F.R. 718.205(c). If the principal cause of death is a medical condition unrelated to pneumoconiosis, the survivor is not entitled to benefits unless the evidence establishes that pneumoconiosis was a substantially contributing cause of the death. 20 C.F.R. 718.205(c)(4).

The Board has held that death will be considered to be due to pneumoconiosis where the cause of death is significantly related to or significantly aggravated by pneumoconiosis. *Foreman v. Peabody Coal Co.,*8 B.L.R. 1-371 (1985). The United States Court of Appeals for the Sixth Circuit, in which the instant case arises, has held that pneumoconiosis is a substantially contributing cause of death if it hastens, even briefly, the miner's death. *See, Brown v. Rock Creek Mining Corp.,* 996 F.2d 812 (6th Cir. 1993)(J. Batchelder dissenting). *See also, Shuff v. Cedar Coal Co.,* 967 F.2d 977 (4th Cir. 1992), *cert. denied,* 113 S.Ct. 969 (1993); *Peabody Coal Co. V. Director, OWCP,* 972 F.2d 178 (7th Cir. 1992); *Lukosevicz v. Director, OWCP,* 888 F.2d 1001 (3rd. Cir. 1989).

The Board has held that in a Part 718 survivor's claim, the Judge must make a threshold determination as to the existence of pneumoconiosis under 20 C.F.R. 718.202(a) before considering whether the miner's death was due to the disease under 718.205. *Trumbo v. Reading Anthracite Co.,* 17 B.L.R. 1-85 (1993). Here, the Employer does not contest the fact that Mr. Baird had pneumoconiosis (Tr. 13).[4] This is amply supported by the conclusions of the physicians who reviewed the medical evidence, conducted the autopsy and treated Mr. Baird, and thus I find that the Claimant has established the existence of pneumoconiosis.

Findings of Fact and Conclusions of Law

I. Background

Mr. Baird was born on October 8, 1942, and died on December 11, 2002 (DX 3). He and the Claimant, Martina Baird, were married on April 10, 1964 (DX 7). Although the Claimant alleged that Mr. Baird worked for at least 28 years as a coal miner, the Director was able to document 15 years of coal mine employment. The Employer has agreed that Mr. Baird worked for at least 15 years as a coal miner, and that it is properly named as the responsible operator. This is supported by Mr. Baird's Social Security earnings report (DX). Thus, I find that Mr. Baird worked as a coal miner for at least fifteen years, and that the Employer is properly named as the responsible operator.

II. Medical Evidence

Dr. Stephen H. Harrison

[4] Nor does the Employer dispute the fact that Mr. Baird's pneumoconiosis arose from his coal mine employment, and I so find.

Dr. Stephen H. Harrison performed the autopsy on Mr. Baird on December 11, 2002 (DX 9). On examination of Mr. Baird's chest, Dr. Harrison noted adhesions involving the left upper and right lower lobes. He found a small, fairly recent thromboembolus in a branch of the right lung. Dr. Harrison noted that the apices of both upper lobes contained ill-defined firm black areas, approximately 5 cm. in greatest dimension each. The left upper lobe was diffusely consolidated, with a very irregular cavity with ragged margins. He noted firm white plaques on the pleural surface at both apices, and numerous firm black nodules throughout all lung fields, ranging up to .8 cm. in greatest dimension. Dr. Harrison found numerous small white plaque-like areas throughout the pleural surface of both lungs; the bronchi were diffusely hyperemic.

On examination of the pleura, Dr. Harrison noted dense fibrous adhesions between the chest wall and left upper and right lower lobes. He found firm white plaques, up to 3 cm. in greatest dimension, on both of the diaphragmatic pleural surfaces.

On microscopic examination of the lungs, Dr. Harrison found recent thrombus focally adherent to a branch of the pulmonary artery. He noted that sections of the left upper lobe showed diffuse acute pneumonia. There was extensive necrosis of alveolar septa, with focal areas of abscess formation. Dr. Harrison also found numerous aggregates of irregular shaped granular blue material, consistent with bacterial colonies, in the abscess cavity. There were large irregularly shaped areas of fibrosis, containing abundant anthracotic pigment, in the apices of both upper lobes. Polarized light microscopy showed numerous birefringent needle like crystals, consistent with silica. There were also a few cavities filled with acellular granular black pigmented material. He noted that the acute pneumonia abutted against the anthracotic focus in the left lobe. Other sections of the lung showed nodular fibrosis with anthracotic pigment and silica particles, as well as emphysematous change.

Dr. Harrison's final diagnoses included anthracosilicosis, with bilateral extensive apical fibrosis, with abundant carbon pigment and silica; bilateral multifocal nodular fibrosis and anthracosis; nodular fibrosis with anthracotic pigment in the hilar lymph nodes; and fibrous pleural plaques. He also found acute lobar pneumonia in the left upper lobe, and a recent pulmonary abscess.

With respect to Mr. Baird's heart, Dr. Harrison noted severe coronary atherosclerosis, with status post myocardial infarction, focal fibrous scar, and compensatory myocardial hypertrophy; and status post coronary artery bypass graft, with a small recent non occlusive thrombosis in the right vein graft. He also found a recent, non occlusive pulmonary thromboembolus.

Dr. Harrison wrote to the Claimant on January 7, 2003, enclosing his autopsy report. He noted that he found extensive changes of anthracosilicosis in both lungs, as well as a severe acute pneumonia involving the left upper lobe, which was complicated by an abscess. According to Dr. Harrison, the pneumonia was the final insult that caused Mr. Baird's death.

Dr. Thomas M. Jarboe

Dr. Jarboe reviewed medical records at the request of the Employer, and prepared a report dated April 24, 2004 (EX 1). However, with the exception of Dr. Harrison's autopsy report, and Mr. Baird's death certificate, the records that Dr. Jarboe reviewed were part of the record in Mr. Baird's living miner claim.

Based on his review, Dr. Jarboe concluded that there was sufficient objective evidence to support a diagnosis of coal workers' pneumoconiosis, based on x-ray interpretations after 1990, as well as Dr. Harrison's autopsy diagnosis. Dr. Jarboe also felt that Mr. Baird had a severe respiratory impairment, in the form of severe obstructive disease. He did not believe that there was sufficient evidence to establish a restrictive ventilatory defect. In Dr. Jarboe's opinion, Mr. Baird's pulmonary impairment was caused by his long history of heavy smoking. He relied on pulmonary function studies that showed marked hyperinflation of the lungs, which is nearly always caused by cigarette smoking, whereas pneumoconiosis causes only mild elevations in residual volume. He also relied on the fact that Mr. Baird had a component of reversible airway disease compatible with asthma; but coal dust inhalation does not cause bronchial asthma. Heavy smoking, however, commonly causes reactive airways disease.

Finally, Dr. Jarboe pointed out that pulmonary function studies showed a pattern of hyperinflation with reduced diffusion capacity and well preserved total lung capacity in 1991, which is classical for pulmonary emphysema. Although pneumoconiosis can be associated with pulmonary emphysema, it is always proportionate to the degree of dust retention in the lungs. However, Mr. Baird's 1990 x-ray showed no nodulation compatible with pneumoconiosis. Therefore, he must have had significant hyperinflation compatible with emphysema before the evolution of radiographic pneumoconiosis. Thus, he concluded that his emphysema was caused by his smoking, and not by his pneumoconiosis. In addition, his normal total lung capacity argued strongly against pneumoconiosis as a cause, as it usually causes at least some element of restriction.

Dr. Jarboe concluded that pneumoconiosis did not have any significant effect on Mr. Baird's course before his death. Indeed, he stated that pneumoconiosis did not cause, contribute to, or in any way hasten his death. He pointed to the autopsy report, which clearly indicated that Mr. Baird died of a severe necrotizing pneumonia. Mr. Baird also had a small pulmonary embolus. In Dr. Jarboe's opinion, this was the cause of his death. Dr. Jarboe acknowledged that Mr. Baird had severe obstructive lung disease, and that his severe airflow obstruction associated with pulmonary emphysema may have contributed to his death. But he felt that his emphysema was caused by his nearly 100 pack year history of smoking, and not by the presence of pneumoconiosis. He knew of no medical literature that supported a causative relationship between pneumoconiosis and an increased incidence of pneumonia. In his opinion, Mr. Baird would have died at the same time and of the same causes whether he had ever worked as a coal miner.

Dr. Jarboe testified by deposition on October 11, 2004 (EX 2). He had been asked to review the reports by Dr. Bush and Dr. Parrish, as well as records from the Methodist Medical Center. Dr. Jarboe acknowledged that Mr. Baird had a significant history of coal dust exposure (22 years), as well as a very strong history of cigarette smoking. He also had a significant history of heart disease.

Dr. Jarboe again acknowledged that Mr. Baird had pneumoconiosis, based on x-ray interpretations and the results of Dr. Harrison's autopsy. However, he did not have complicated pneumoconiosis. Dr. Jarboe pointed out that Dr. Harrison described firm black areas five centimeters in greatest diameter, but that description does not indicate complicated pneumoconiosis. According to Dr. Jarboe, there must be microscopic findings of active inflammation around the edges of the lesions. But Dr. Harrison did not describe rounded masses in the chest, and the lesions could have been linear depositions of coal dust, which is simply dust deposition, or an area of atelectasis. Thus, Dr. Harrison's description did not allow for a diagnosis of complicated pneumoconiosis.

Again, Dr. Jarboe stated that Mr. Baird died of a severe necrotizing pneumonia, but his death was not caused by, related to, or hastened by pneumoconiosis or exposure to coal dust. He once more noted his primary reason, that there was no evidence in the medical literature that pneumoconiosis makes a person susceptible to pneumonia, specifically bacterial pneumonia. He also felt that for pneumoconiosis to have contributed to Mr. Baird's death, it would have had to cause significant respiratory impairment. While Mr. Baird had respiratory impairment, in his opinion it was caused by his long and very heavy history of smoking, and not by the presence of pneumoconiosis in his lung tissues.

Dr. Jarboe disagreed with Dr. Parrish, who concluded that Mr. Baird had complicated pneumoconiosis, and that his death was due to complications thereof. In Dr. Jarboe's opinion, Mr. Baird died of a necrotizing pneumonia, unrelated to the presence of simple pneumoconiosis in his lungs. According to Dr. Jarboe,

> [Mr. Baird] had significant obstructive lung disease and pulmonary emphysema. That could have contributed to or made him more susceptible to the presence of pneumonia. That's a generally accepted clinical axiom. But in my opinion, as I have just gone over in detail, I don't think that the obstructive lung disease was caused by the inhalation of coal dust or the presence of pneumoconiosis in his pulmonary tissues. So I don't think that pneumoconiosis caused or substantially contributed to his death.

(EX 2 at p. 22).

Dr. Lawrence Repsher

Dr. Repsher reviewed medical records at the request of the Employer, and prepared a report dated April 26, 2004 (EX 3). Dr. Repsher previously prepared reports in connection with Mr. Baird's living miner's claim, dated March 9, 1999 and September 22, 1999; he also reviewed Dr. Harrison's autopsy report, as well as his letter to the Claimant. From Mr. Baird's living miner's exhibit file, he reviewed additional reports that he had not previously seen.

Dr. Repsher concluded that there was sufficient objective evidence to justify a diagnosis of simple pneumoconiosis. He also felt that Mr. Baird had a severe pulmonary and respiratory impairment, but that it could not be attributed to pneumoconiosis. Rather, it was due solely to his long and heavy smoking habit. He stated that simple pneumoconiosis, on the average, does

not cause any clinically significant impairment of lung function. Thus, "to an overwhelming statistical probability, his COPD was due exclusively to an individually measurable extent to his cigarette smoking habit and was not related to any individually measurable extent to his work as a coal miner with exposure to coal mine dust."

In Dr. Repsher's opinion, pneumoconiosis did not play any significant role in Mr. Baird's clinical course before death. He stated that Mr. Baird's pneumoconiosis did not involve enough lung tissue to cause any individually measurable reduction in his breathing capacity.

Dr. Repsher testified by deposition on May 3, 2004, after reviewing the reports by Dr. Naeye and Dr. Parrish (CX 4). Dr. Repsher noted that Dr. Harrison, who performed the autopsy, did not find histologic evidence of complicated pneumoconiosis. According to Dr. Repsher, if a person had complicated pneumoconiosis, a competent autopsy would show evidence of it.

Dr. Repsher disagreed with Dr. Parrish's conclusion that Mr. Baird's pneumonia was related to pneumoconiosis. He also noted that Dr. Parrish found that Mr. Baird had complicated pneumoconiosis, without explanation, when two pathologists did not find it. According to Dr. Repsher, pneumoconiosis, or the inhalation of coal dust, does not impair any of the defenses against bacterial infection or bacterial pneumonia.

Dr. Richard L. Naeye

Dr. Naeye reviewed medical records at the request of the Employer, and also examined Mr. Baird's autopsy slides (EX 5). The records, with the exception of the autopsy report, were part of Mr. Baird's file in his living miner's claim.

Dr. Naeye noted that seven of the autopsy slides contained lung tissue, and four contained heart tissue. Although the slides were very poorly stained, he felt that their tissue findings could be interpreted. According to Dr. Naeye, cross sections of coronary arteries showed severe or very severe arteriosclerosis, but no hyalinized collagen, which could be interpreted as old microinfarcts.

Dr. Naeye's examination of the lung tissue showed old, hyalinized collagen with admixed anthracotic pigment and many birefringent crystals of all sizes at multiple sites adjacent to small arteries and airways, and below the pleura. He indicated that at three sites these lesions reached or exceeded 1 cm. in diameter. At some sites in the lesions, there were large numbers of birefringent crystals, some of which were very tiny, and were crystals of toxic free silica. There was only a small amount of black pigment associated with these lesions, so they were very old. At a few sites, there was mild or moderate centrilobular emphysema, and acute lobular pneumonia that had reached the abscess stage.

According to Dr. Naeye, the records made it clear that Mr. Baird had very severe coronary artery disease and obstructive disease, the latter due to his more than sixty pack year history of smoking. This was the major cause of his late life disability and death. Dr. Naeye stated that Mr. Baird's pulmonary function studies showed that his lung disease was never disabling; however, lung disease in the form of pneumonia in the abscess stage was the direct

cause of his death. According to Dr. Naeye, there were findings of pneumoconiosis at death in the form of very old anthracotic silicotic lesions, two of which just reached 1 cm. in greatest dimension. The centers were usually necrotic. The lesions appeared inactive; their edges did not show fibroblastic activity or sizable numbers of chronic inflammatory cells. Thus, they had not grown for at least several years before Mr. Baird died. Because of this inactivity, they did not meet the minimum criteria for a diagnosis of complicated coal workers' pneumoconiosis.

Dr. Naeye testified by deposition on May 14, 2004 (EX 5). According to Dr. Naeye, originally a pathologic diagnosis of complicated pneumoconiosis pathologically included lesions, with chronic inflammatory cells, fibroblasts, and recently deposited fibrin at the edge. The lesion has the characteristic feature of damaging blood vessels, so the center, when the lesions become large, are always necrotic. Dr. Naeye did not diagnose complicated coal workers' pneumoconiosis, stating that to do so, there must be a lesion of two centimeters in tissue measurement. He stated that a lesion that appears as one centimeter on x-ray will be much larger in tissue, because at the edge of the lesion, the x-rays will pass through without causing a shadow.

According to Dr. Naeye, there are also cases involving a silicotic disorder in which layers and layers of fibrosis are laid down on the outer regions of the lesion, as the tiny crystals of free silica migrate. If the lesion reaches two centimeters, it is widely accepted as another definition of complicated pneumoconiosis.

Dr. Naeye felt that Mr. Baird's history of smoking had an impact on his pulmonary system, as well as his cardiac system. He noted that clinically, Mr. Baird had evidence of airway obstruction in testing before 1994. According to Dr. Naeye, cigarette smoking has a five to seven times greater influence than exposure to coal mine dust. Because Mr. Baird's arterial blood gas results were normal until 1994, he felt there was no significant evidence of emphysema. In Dr. Naeye's opinion, Mr. Baird's progressive dyspnea on exertion was due to his cardiac damage, but his bronchitis may also have contributed.

In his review of the autopsy slides, Dr. Naeye found evidence that Mr. Baird had been exposed to free silica damage. At three sites, there were lesions of one centimeter in greatest dimension, with features indicating that free silica had caused damage: there was deep fibrosis with admixed amounts of very tiny birefringent toxic crystals. Overall, however, emphysema, mainly centrilobular, was only mild to moderate. Dr. Naeye felt that Mr. Baird did not have enough emphysema to have impaired his lung function. His problem was mainly bronchitis and bronchiolitis.

According to Dr. Naeye, smoking has a much greater effect in causing bronchitis than does coal mine dust, although "one potentiates the other." In other words, if someone who smokes develops chronic bronchitis, it does not go away after ceasing coal mining. Whereas, in a non smoker who develops industrial bronchitis while mining coal, it will almost always disappear after he quits mining coal.

According to Dr. Naeye, the primary cause of Mr. Baird's death was the very severe damage in his coronary circulation. But he also had very severe obstructive lung disease due to

his cigarette smoking. Dr. Naeye stated that although smoking has a much greater effect than coal mine dust, mine dust does have a minor effect. As he noted in his report, Dr. Naeye stated that lung disease in the form of pneumonia was the direct cause of death. He noted that cigarette smoking tremendously damages the defense mechanisms in the lungs, increases the likelihood of developing pneumonia, and makes recovery much more difficult. He disagreed with Dr. Parrish's statement that Mr. Baird's pneumonia was the direct result of pneumoconiosis. He stated that while damage due to pneumoconiosis can have some effect on defense mechanisms in the lung, it is very small by comparison with the effects of cigarette smoking. He felt that the effects of cigarette smoking were the dominating problem. According to Dr. Naeye, Mr. Baird's death was not caused by, related to, or hastened in any significant way by his occupational exposure to coal dust.

Dr. Stephen T. Bush

Dr. Bush reviewed medical records at the Employer's request, and prepared a report dated March 23, 2004 (EX 6). With the exception of Mr. Baird's death certificate, Dr. Harrison's autopsy report and letter, Dr. Naeye's report, and the eleven autopsy slides, all of the records were from Mr. Baird's living miner's claim file.

Dr. Bush concluded that the medical records and histologic slides provided sufficient objective evidence to support a diagnosis of a moderate degree of pneumoconiosis. He noted that four of the slides showed micronodules up to .5 cm., consisting of dust pigment associated with a dense fibrous reaction, forming nodules surrounded by minimal focal emphysema. Polarized light examination showed a large number of birefringent particles, consistent with silica and silicates; dust pigment was relatively small in quantity. Three of the slides showed more profuse nodularity by similar lesions, with large quantities of silica and central areas of degeneration. The lesions were 1 cm. in smallest dimension, but did not contain the deeply pigmented, exuberant, destructive fibrosis of progressive massive fibrosis.

According to Dr. Bush, each slide showed, to a greater or lesser extent, necrotizing bronchopneumonia, with only outlines of alveoli filled with fibrin and some inflammatory cells. He noted fibrous thickening of the pleura in several areas, associated with only a small amount of dust pigment. He stated that centrilobular emphysema was locally moderate but generally mild, although the degree could be obscured by the extensive alveolar fluid with fibrin.

Dr. Bush reported that considering the sparse distribution of coal worker changes in the lung sections other than the apical regions, the lesions affected only a few percent of the lung substance; when the apical areas were considered, the lung destroyed by pneumoconiosis totaled an estimated ten to fifteen percent. According to Dr. Bush, this is a moderate degree of pneumoconiosis, which is not consistent with disabling disease.

Dr. Bush concluded that Mr. Baird had significant respiratory impairment, repeatedly confirmed as obstructive disease due to his long and heavy cigarette smoking history. In Dr. Bush's opinion, his pulmonary impairment was not of the restrictive or mixed type typically found in pneumoconiosis. He noted that none of the cardiac evaluations suggested an abnormality of the right ventricle that might be expected with severe chronic pulmonary disease

such as pneumoconiosis. He also noted that Mr. Baird's non-specific complaints of shortness of breath could have arisen from his chronic cardiac failure.

In Dr. Bush's opinion, Mr. Baird's impairment was a result of a severe obstructive defect, which was the result of chronic obstructive pulmonary disease related to cigarette smoking. According to Dr. Bush, pneumoconiosis had no significant effect on his course before death, as it was too limited in degree and extent to have made any significant contribution to his medical problems.

According to Dr. Bush, pneumoconiosis did not cause, contribute to, or in any way hasten Mr. Baird's death, and he would have died in the same manner and at the same time if he had no coal workers' disease. He died of massive bronchopneumonia with pulmonary edema associated with severe coronary artery disease. His death would not have been prevented or delayed if he had never been exposed to coal mine dust.

Dr. Richard E. Parrish

Dr. Parrish wrote a letter on the Claimant's behalf, dated March 26, 2003 (DX 11). He stated that he had treated Mr. Baird for ten years, and had maintained since 1992 that he had significant coal workers' pneumoconiosis. He enclosed a letter to Judge Mollie Neal, documenting his belief that Mr. Baird had progressive massive fibrosis. He noted that Mr. Baird's autopsy was consistent with complicated pneumoconiosis and anthracosis. According to Dr. Parrish, there was no doubt that Mr. Baird had obstructive lung disease and pulmonary emphysema to a degree. But he noted that Dr. Harrison's report did not include any findings consistent with emphysema; all of the findings were related to pneumoconiosis and pneumonia, which Dr. Parrish felt to be a direct result of Mr. Baird's pneumoconiosis. Dr. Parrish reiterated that, as he indicated on Mr. Baird's death certificate, his death was related to complications of pneumoconiosis.

Dr. Parrish testified by deposition on April 30, 2004 (EX 7). He described complicated pneumoconiosis as larger macules in the lungs, with coalescence and hilar retractions in the upper lobes. According to Dr. Parrish, that was Mr. Baird's condition. Dr. Parrish diagnosed Mr. Baird with coal workers' pneumoconiosis and COPD. He explained that people do not have pure emphysema or pure chronic bronchitis, but they usually have some of both. He felt that Mr. Baird probably had some of each, although he noted that his autopsy report did not show emphysema.

According to Dr. Parrish, he diagnosed pneumoconiosis based first on Mr. Baird's history of coal mine employment, about ten years of which was underground. In addition, his x-rays showed abnormalities that indicated complicated pneumoconiosis. Mr. Baird was also short of breath. Dr. Parrish prescribed bronchodilators, and antibiotics and steroids for infections. He noted that treatments for pneumoconiosis and COPD were not very satisfying, as the conditions do not respond very well to treatment, but these are all they have, and they attempt to treat it as best they can.

Dr. Parrish felt that the findings in Dr. Harrison's autopsy report were consistent with complicated pneumoconiosis. He noted extensive fibrosis with carbon pigment in both lungs. Dr. Parrish felt that Mr. Baird's pneumonia was the direct result of pneumoconiosis, and that pneumonia is usually seen in people with underlying diseases. Mr. Baird's was severe. According to Dr. Parrish, the way most people get pneumonia is by breathing in bacteria. Usually, a body can fight these off with various defense mechanisms. But people with diseased lungs do not have the defense mechanisms of healthy lungs, making it more likely for pneumonia to develop.

Dr. Parrish acknowledged that assessment of a person's smoking history was important in determining the cause of impairment, as smoking does cause obstructive lung disease. In Mr. Baird's case, he thought there were two things that caused his lung impairment: his work exposure, and his cigarette smoking. He noted that the autopsy did not mention emphysema, which is usually caused by smoking. According to Dr. Parrish, pneumoconiosis can cause obstructive, restrictive, or a combination of abnormalities. Cigarette smoking usually causes obstructive abnormalities.

Dr. Parrish stated that, although the autopsy did not show it, he thought Mr. Baird had emphysema clinically. He described the autopsy as the ultimate diagnostic test, which is always one hundred percent accurate. In his view, the autopsy showed that as a cause of Mr. Baird's COPD, pneumoconiosis predominated.

Dr. Parrish completed Mr. Baird's death certificate, listing as the immediate cause of death respiratory failure, due to pneumonia and coal workers' pneumoconiosis (DX 8). Dr. Parrish also indicated that COPD and coronary artery disease were also significant conditions contributing to Mr. Baird's death.

The record also includes a letter from Becky Cunningham, a nurse in Dr. Parish's office, dated January 16, 2003 (CX 1). Ms. Cunningham indicated that Mr. Baird had been seen in their office for coal workers' pneumoconiosis and COPD, and admitted to the hospital on December 2, 2002 for exacerbation of his respiratory problems. While in the hospital, he experienced increased shortness of breath, and was transferred to the ICU with impending respiratory failure.

Methodist Medical Center

The record includes the hospitalization records for Mr. Baird's December 2, 2002 admission (CX 2). The discharge summary was prepared by Dr. Parrish, who stated that he had known Mr. Baird for a number of years. According to Dr. Parrish, Mr. Baird had significant chronic obstructive pulmonary disease, and coal workers' pneumoconiosis with progressive massive fibrosis in both upper lungs. Mr. Baird had a past history of coronary artery disease, and he underwent coronary artery bypass grafting and stenting earlier that year.

On his initial examination of Mr. Baird, Dr. Parrish noted that he was wheezing and there were retractions. The cardiac exam showed tachydardia. His chest x-ray showed bilateral upper lobe infiltrates, consistent with complicated coal workers' pneumoconiosis. Mr. Baird was admitted and treated with steroids, antibiotics, and respiratory therapy, but he developed

worsening respiratory distress and required intubation in the ICU. He developed multiorgan failure and progressive shock from sepsis, and died on December 11, 2002.

During the course of Mr. Baird's final hospitalization, several chest x-rays were taken. A radiology report by James Parrott dated December 6, 2002 states:

> Question chronic obstructive lung disease with extensive stranding in both upper lobes, elevation of the hila suggesting fibrotic lung disease.

Dr. William Prater read a chest x-ray on December 10, 2002, noting some interval increase in parenchymal infiltrate in the right upper lung, superimposed on chronic parenchymal scarring. His impression was increasing parenchymal density in the right upper lung.

In an x-ray study for the purpose of determining placement of a tube on December 10, 2002, Dr. Peter G. Emanuel noted infiltrates in the right upper lobe, and more diffuse left sided infiltrates.

On December 11, 2002, Dr. Parrott read an x-ray to determine tube placement, noting marked densities in the superior half of the right lung, and diffuse haziness throughout the left lung field. He indicated that these findings were more marked than on December 10.

Dr. Parrish wrote to Judge Neal on March 13, 2002 regarding Mr. Baird's current medical condition (DX 1). He indicated that he believed that Mr. Baird had complicated coal workers' pneumoconiosis; he referred to a 1999 x-ray that showed bilateral nodular pulmonary fibrosis, and his pulmonary function studies at that time. According to Dr. Parrish, Mr. Baird had been hospitalized for pulmonary complaints, which Dr. Parrish felt were related to his pneumoconiosis.

Dr. Parrish stated that Mr. Baird's x-rays now showed large conglomerate lesions in both upper lung fields with retraction of the hila, which he would rate as category C opacities under the ILO classification. Mr. Baird's pulmonary function studies since 1999 had also deteriorated, and his symptoms had worsened; he was short of breath with minimal exertion.

Dr. Parrish stated his opinion that Mr. Baird had complicated coal workers' pneumoconiosis, with progressive radiographic changes and decline in his pulmonary function tests. He felt that Mr. Baird's pulmonary impairment was due in large3 part to this complicated pneumoconiosis, from his coal mine employment.

Dr. Parrish attached a letter to Judge Neal dated September 9, 1999, again regarding Mr. Baird's current medical condition. He indicated that he had treated Mr. Baird since 1994, when he was referred by his family physician. He noted Mr. Baird's 15 to 16 year history of coal mine employment, at least 10 of which were underground. Dr. Parrish stated that Mr. Baird had a history of coronary artery disease, and had had coronary artery bypass grafting. He smoked, but quit in 1989.

According to Dr. Parrish, Mr. Baird's chest x-ray was consistent with complicated pneumoconiosis. He noted that B readers had documented the fact that he has bilateral nodular pulmonary fibrosis in both upper lung fields, consistent with complicated pneumoconiosis. Mr. Baird's pulmonary function tests showed severe obstructive impairment, with his most recent FEV1 being 28% of predicted.

Dr. Parrish acknowledged that cigarette smoking had contributed to Mr. Baird's impairment, but he believed that his industrial exposure to coal dust, and his x-ray evidence of pulmonary fibrosis were also related to his pulmonary impairment. Attached was an interpretation by Dr. Parrish of an x-ray taken on December 2, 1999, showing markedly hyperexpanded lungs, and evidence of bilateral fibrosis in both upper lung fields consistent with coal workers' pneumoconiosis.

Attached to Dr. Parrish's September 9, 1999 letter were treatment records and x-ray reports. Dr. Parrish interpreted an x-ray taken on June 9, 1999, noting bilateral upper lobe fibrotic conglomerate lesions consistent with coal workers' pneumoconiosis. Dr. Manley Jordan reviewed an x-ray taken February 24, 2000, noting increased hyperinflation, and anthracosilicosis complicated by conglomerate formation with ventricular nodular changes in the mid upper lung zones. He noted a new infiltrate in the right lower lobe, and could not exclude pneumonia.

Dr. Parrish read Mr. Baird's December 7, 2000 x-ray, noting evidence of coal workers' pneumoconiosis with PMF lesions in both upper lung fields.

Upon reading an x-ray dated September 18, 2000, Dr. Parrish noted marked hyperexpansion, and bilateral fibrosis with PMF in the upper lobes, consistent with coal workers' pneumoconiosis.

Dr. Jordan reviewed an x-ray dated April 4, 2000, noting changes of complicated pneumoconiosis, with conglomerate formation in both apices, with irregular-rounded opacities.

Dr. Parrish wrote a letter dated July 13, 1995 on Mr. Baird's behalf (DX 1). He noted that his office had been treating Mr. Baird for the last year; he had a 15 to 16 year history of coal mining, at least ten of those underground. He also smoked from age ten until December 199. Recent pulmonary function tests were consistent with a severe obstructive defect.

In Dr. Parrish's opinion, coalworkers' pneumoconiosis was contributing to Mr. Baird's lung disease and disability. Dr. Parrish acknowledged that smoking had a lot to do with the development of obstructive lung disease, but he also felt that pneumoconiosis was playing a part, although he could not say with certainty that one or the other was exclusively responsible. But given Mr. Baird's work history, smoking history, x-ray findings, and pulmonary function test results, he felt that his coal working experience and documented black lung certainly was a contributing factor to the development of disability related to his lung disease.

In a letter dated November 10, 1994, Dr. Parrish reported that he had seen Mr. Baird for a black lung evaluation. A chest x-ray read by a board certified radiologist in July 1994 showed

changes of 2/3, consistent with pneumoconiosis by ILO classification. Mr. Baird's pulmonary function study results were also consistent with a severe obstructive impairment. In Dr. Parrish's opinion, Mr. Baird's lung condition was related to his work in the coal mines.

The Claimant's representative, Ms. Hutson, also designated a "letter of support" from Dr. Parrish dated May 24, 1994. However, I have been unable to locate any such letter in the exhibit file. There is a report by Dr. Parrish dated May 23, 2994, which discusses Dr. Parrish's initial examination of Mr. Baird, on referral from Dr. Goff. Essentially, Dr. Parrish makes the same conclusions as in his November 10, 1994 letter.

DISCUSSION

For the reasons discussed below, I find that the Claimant has established by a preponderance of the reliable and admissible medical evidence that Mr. Baird's respiratory death was due to coalworkers' pneumoconiosis. In this regard, the United States Court of Appeals for the Third Circuit has held that any condition that *hastens* the miner's death is a substantially contributing cause of death for purposes of §718.205. *Lukosevicz v. Director, OWCP*, 888 F.2d 1001 (3rd Cir. 1989). The Fourth, Sixth, Seventh, and Tenth Circuits have adopted this position in *Shuff v. Cedar Coal Co.*, 967 F.2d 977 (4th Cir. 1992), *cert. denied*, 113 S. Ct. 969 (1993); *Brown v. Rock Creek Mining Corp.*, 996 F.2d 812 (6th Cir. 1993)(J. Batchelder dissenting*);* and *Peabody Coal Co. v. Director, OWCP*, 972 F.2d 178 (7th Cir. 1992); *Northern Coal Co. v. Director, OWCP*, 100 F.3d 871 (10th Cir. 1996) (a survivor is entitled to benefits if pneumoconiosis hastened the miner's death "to any degree").

Issue Preclusion

There is no dispute that Mr. Baird had coalworkers' pneumoconiosis. Nor is there any dispute that he suffered from disabling chronic obstructive pulmonary disease. Thus, Dr. Jarboe acknowledged that Mr. Baird had severe obstructive lung disease, and further that this severe airflow obstruction may have contributed to his death, or made him more susceptible to the pneumonia that was the direct cause of his death. Dr. Repsher also acknowledged that Mr. Baird had a severe obstructive pulmonary and respiratory impairment. It was also clear to Dr. Naeye that Mr. Baird had very severe obstructive disease that was the major cause of his disability and death. Finally, Dr. Bush concluded that Mr. Baird had significant obstructive respiratory impairment.

To this extent, these opinions are consistent with those of Mr. Baird's treating physician, Dr. Parrish, a pulmonary specialist who treated Mr. Baird for almost ten years for chronic obstructive pulmonary disease as well as pneumoconiosis. They are also consistent with the conclusion that Mr. Baird's severe obstructive disease, while not the direct cause of his death, contributed to or hastened that death by pneumonia. However, all of these physicians go to great lengths to exclude Mr. Baird's pneumoconiosis as a factor of any kind in his obstructive disease, and thus break the causal chain between his pneumoconiosis and his pulmonary death.

But this issue has already been resolved. In her Decision and Order on Remand, issued on November 3, 2002, Judge Neal reconsidered the issue of whether pneumoconiosis was a substantially contributing cause of Mr. Baird's total disability.[5] Judge Neal discussed the medical evidence thoroughly, including the opinions by Dr. Repsher, and concluded that the most probative opinion regarding the cause of the Claimant's totally disabling respiratory impairment was that of Dr. Parrish. Judge Neal stated:

> Dr. Parrish is a qualified pulmonary specialist. He has treated the Claimant for over six years for his respiratory impairment and coal workers' pneumoconiosis. . . . his treatment notes document that he has regularly examined the miner on follow up visits, monitored his condition by diagnostic tests (pulmonary function studies and chest x-rays), and prescribed a course of medication therapy. His opinion is documented by the objective medical evidence and satisfies the requirement for credible evidence. His opinion is further consistent with the underlying purposes of the Act, in that it is [sic] incorporates the broad definition of coal workers pneumoconiosis, and recognizes the irreversible progressive nature of the disease process. Dr. Parrish attributes the miner's disability to the risk factors of smoking and coal dust exposure. While he cannot definitively state the degree to which either factor contributes to the miner's respiratory disability, his inability to do so does not affect the weight which can be given to his opinion. The courts have recognized that the question of the relative amounts that various causal elements contribute to a totally disabling respiratory impairment can be extremely problematic. *See Adams,* 886 F.2d 825; *Cross Mountain Coal Co. v. Ward,* 93 F.3d 211, 218 (6th 1996); *Compton v. Inland Steel Coal Co.,* 933 F.2d 477, 481-483 (7th Cir. 1991).

Decision and Order at p. 15. Although the Employer appealed Judge Neal's decision to the Benefits Review Board, after Mr. Baird died, it requested that the Board dismiss its appeal. The Board issued its Order dismissing the Employer's appeal on February 28, 2003.

The Board, in an unpublished decision, has held that the doctrine of collateral estoppel precluded an employer in a widow's claim from relitigating the determination made almost two years earlier in the miner's claim that he had pneumoconiosis. *Young v. Sewell Coal Co.,* BRB No. 98-1000 BLA (Aug. 26, 1999). In that case, the Board stated:

> Collateral estoppel forecloses "the relitigation of issues of fact or law that are identical to issues which have been actually determined and necessarily decided in prior litigation in which the party against whom [issue preclusion] is asserted had a full and fair opportunity to litigate." *Ramsey v. INS,* 14 F.3d 206 (4th Cir. 1994); *see Virginia Hosp. Ass'n v. Baliles,* 830 F.2d 1308 (4th Cir. 1987); *see also Freeman v. [sic]; United Coal Mining Co. v. Director, OWCP [Forsythe],* 20 F.3d 289, 18 BLR 2-189 (7th Cir. 1994). For collateral estoppel to apply in the present case, which arises within the jurisdiction of the United States Court of Appeals for the Fourth Circuit, claimant must establish that:
>
> (1) the issue sought to be precluded is identical to one previously litigated;
> (2) the issue was actually determined in the prior proceeding;

[5] Indeed, this was the only issue before Judge Neal on remand.

(3) the issue was a critical and necessary part of the judgment in the prior proceeding;
(4) the prior judgment is final and valid; and
(5) the party against whom estoppel is asserted had a full and fair opportunity to litigate the issue in the previous forum.

See Sedlack v. Braswell Services Group, Inc., 134 F.3d 219 (4th Cir. 1998); *Sandberg v. Virginia Bankshares, Inc.,* 979 F.2d 332 (4th Cir. 1992); *Ramsey, supra.*

Young v. Sewell Coal Co., supra, slip op. at 4. The Seventh Circuit Court of Appeals has affirmed the application of collateral estoppel for findings from a miner's claim to a widow's claim. *Zeigler Coal Co. v. Director, OWCP [Villain],* 312 F.3d 332 (7th Cir. 2002). However, the Seventh Circuit (as well as the Board) has recognized an exception for cases where autopsy evidence is first introduced in the widow's claim on the issue of the existence of pneumoconiosis, noting that autopsy evidence is in essence the gold standard for determining the existence of pneumoconiosis, and the Employer is entitled to submit evidence on the basis of autopsy results that were not available in the miner's claim.

Here, the very narrow issue of whether Mr. Baird's disabling obstructive pulmonary condition was due to pneumoconiosis is identical in both claims. In the miner's claim, Judge Neal made a determination that Mr. Baird's disabling obstructive pulmonary condition was in fact due to pneumoconiosis, a finding that was a critical and necessary part of the award of benefits. Judge Neal's determination was not appealed, and thus has become final. Key Mining Incorporated was the designated responsible operator in Mr. Baird's miner's claim, and fully litigated that issue. Key Mining Incorporated had the opportunity to appeal Judge Neal's determination on that issue, but chose not to do so.

I find that all of the conditions for issue preclusion have been met, and that Judge Neal's finding that Mr. Baird's totally disabling obstructive pulmonary impairment was due to pneumoconiosis is binding in the Claimant's widow's claim.

Finally, I find that the "autopsy evidence" exception to the issue preclusion rule does not apply in this situation. That exception recognizes that on the question of whether a miner had pneumoconiosis, autopsy evidence is the most reliable, and as that evidence is customarily not available in a miner's claim, applying the issue preclusion rule would prevent the Employer from fully litigating that issue in the widow's claim. Here, however, the physicians upon whose opinions the Employer relied did not base their conclusions on the narrow issue of the etiology of Mr. Baird's obstructive impairment on the autopsy evidence. Rather, they based their opinions on the results of pulmonary function studies and other clinical testing performed on Mr. Baird when he was alive, the very evidence that was considered in the miner's claim, on an issue that the Employer has had more than ample opportunity to litigate.

In Mr. Baird's living miner's claim, the Employer chose not to pursue an appeal on the very issue squarely presented here: whether Mr. Baird's disabling obstructive disease was due, at least in part, to his pneumoconiosis. The Employer should not be allowed to re-open that same issue here, relying on the same medical evidence that was before Judge Neal.

Evidentiary Limitations

The Employer has submitted and designated the reports of Dr. Jarboe and Dr. Repsher as its two initial medical reports. In each of these reports, the physician has reviewed medical evidence that is part of the Claimant's file, as well as the medical evidence that was part of Mr. Baird's living miner's claim. I informed the parties at the beginning of the hearing that, as required by the regulations, I was offering into the record all of the Director's exhibits (which included Mr. Baird's living miner's claim), ***with the caveat that they were subject to the limitations of the new regulations*** (Tr. 5).

The Board has found that in cases under the new regulations, medical data that underlies a medical report must itself be admissible. *Dempsey v. Sewell Coal Co.*, __ B.L.R. __, BRB Nos. 03-0615 BLA and 03-0615 BLA-A (June 28, 2004) (en banc). In that case, the Board found that the Administrative Law Judge properly declined to consider a report admitted as part of the Employer's affirmative case, in which a physician provided a medical opinion based in part on his interpretation of a chest x-ray study that was not part of the record.

The Employer did not specifically designate any of the voluminous medical records from the file generated in connection with Mr. Baird's living miner's claim as exhibits in this claim. Thus, in its Black Lung Benefits Act Evidence Summary Form submitted on October 18, 2004, the Employer designated as its two initial medical reports the reports and deposition testimony of Dr. Repsher and Dr. Jarboe; the Employer designated Dr. Naeye's autopsy report and deposition as its initial autopsy report, and Dr. Bush's report as its rebuttal autopsy report. The Employer did not designate or even offer any of the medical evidence from Mr. Baird's living miner's file, despite the statement on the evidence summary form that "medical reports" may only be based on medical evidence which is admissible consistent with the evidentiary limitations (ALJX 4).

It is clear from Dr. Repsher's report and testimony that he reviewed and relied not only on the medical evidence in the Claimant's file, but also all of the medical evidence generated in connection with Mr. Baird's living miner's claim. Indeed, he incorporated his previous report of March 9, 1999, and a letter dated September 22, 1999, and reviewed additional reports generated in connection with Mr. Baird's living miner's claim. Not only were these medical records not a part of the record here, they far exceed the evidentiary limitations under the new regulations. Nor did the Employer even proffer any basis for an exception of good cause. Dr. Jarboe's lengthy report makes it clear that he also reviewed and relied upon all of the evidence in Mr. Baird's living miner's claim in making his conclusions.

Although the Employer designated the reports of Dr. Bush and Dr. Naeye as "autopsy evidence," their reports are much more expansive, and in addition to their review of the autopsy findings and slides, consider and discuss the records from the Claimant's claim, as well as from Mr. Baird's living miner's claim. Both Dr. Bush and Dr. Naeye relied on their review of these medical records to expound on their assessment of Mr. Baird's respiratory or pulmonary condition when he was alive. Their reports exceed the bounds of a report on autopsy findings, and are more properly characterized as a combination of autopsy reports and medical reports. But the Employer has already submitted medical reports from Dr. Repsher and Dr. Jarboe as the

two medical reports allowed under the regulations. Thus, not only do Dr. Bush's and Dr. Naeye's reports impermissibly rely on medical evidence that is not part of the record, to the extent that they expound on issues outside of a review of the autopsy findings or slides, they become medical reports, which impermissibly exceed the Employer's evidentiary limitations.

In any event, the opinions of Dr. Jarboe, Dr. Repsher, Dr. Bush, and Dr. Naeye on the crucial issue here – whether Mr. Baird's death was due to pneumoconiosis – are so clearly based on and inextricably intertwined with their assessment of medical records that are not properly part of this record, that I find that I cannot place any reliance on their opinions.

Merits of the Claim

Dr. Parrish treated Mr. Baird for ten years before his death. He completed Mr. Baird's death certificate, and listed respiratory failure as the immediate cause of death, due to pneumonia and coal workers' pneumoconiosis (DX 8). He also stated that COPD and coronary artery disease were significant conditions contributing to his death.

In his deposition, Dr. Parrish testified that he had diagnosed Mr. Baird with coal workers' pneumoconiosis and COPD, based on his history of coal mine employment, his x-rays, and his shortness of breath. Dr. Parrish treated Mr. Baird with bronchodilators, antibiotics, and steroids. He reviewed Dr. Harrison's autopsy report, noting the extensive fibrosis with carbon pigment in both lungs. According to Dr. Parrish, Mr. Baird had severe underlying pulmonary disease, and pneumonia is usually seen in such people, who breathe in bacteria, but do not have the defense mechanisms of healthy lungs to fight the bacteria. Thus, pneumonia is more likely to develop.

Dr. Parrish acknowledged that smoking can cause obstructive lung disease, and should be considered when determining the cause of lung impairment. In Dr. Parrish's opinion, Mr. Baird's lung impairment was due to his work exposure as well as his history of cigarette smoking. Because the autopsy did not show emphysema, even though Dr. Parrish felt that Mr. Baird had clinical symptoms, Dr. Parrish stated that pneumoconiosis predominated as the cause of Mr. Baird's chronic obstructive pulmonary disease.

The new regulations at 20 C.F.R. § 718.104, which codify judicial precedent, provide that significant weight may be given to the opinion of a treating physician. As Dr. Parrish was Mr. Baird's treating physician, I find that his opinion is entitled to great weight. Dr. Parrish's resume reflects that he is a well-qualified pulmonary specialist. He discussed the basis for his diagnosis of pneumoconiosis, and his conclusion that Mr. Baird's obstructive lung disease was due to a combination of his exposure to coal mine dust and his cigarette smoking, and referred to his x-ray and pulmonary function study findings, and Mr. Baird's clinical symptoms. He also relied on the autopsy findings, which confirmed his conclusion that pneumoconiosis was a significant factor in Mr. Baird's obstructive impairment. I agree with Judge Neal, that while Dr. Parrish cannot definitively state the degree to which either smoking or coal dust exposure contributed to Mr. Baird's respiratory disability, this does not affect the weight that can be given to his opinion, as the courts have recognized that the question of the relative amounts that various causal elements contribute to a totally disabling respiratory impairment can be extremely problematic. *See, Adams v. Director, OWCP*, 886 F.2d 818, 825 (6th Cir. 1989); *Cross Mountain Coal Co. v.*

Ward, 93 F.3d 211, 218 (6th Cir. 1996); *Compton v. Inland Steel Coal Co.,* 933 F.2d 477, 481-483 (7th Cir. 1991).[6]

Dr. Harrison, the autopsy prosector, addressed the issue of the contribution of pneumoconiosis to Mr. Baird's death only obliquely. Thus, in his letter to the Claimant forwarding a copy of his autopsy report, he noted that he found extensive changes of anthracosilicosis in both lungs, and a severe acute pneumonia in the left upper lobe, which was complicated by an abscess. He told the Claimant that pneumonia was the "final insult" that caused Mr. Baird's death. I interpret this statement as indicating that pneumonia, along with the extensive changes of anthracosilicosis noted by Dr. Harrison, combined to result in Mr. Baird's death. While I find that Dr. Harrison's comment may not be sufficiently unequivocal in itself to support a finding that Mr. Baird's pneumoconiosis hastened his death, I find that it supports Dr. Parrish's conclusions; it certainly does not detract from them.

Even if I were to consider the opinions of Dr. Naeye and Dr. Bush as "autopsy reports," I find them to be contradictory, confusing, and equivocal. Thus, Dr. Naeye stated that Mr. Baird's death was not caused by, related to, or hastened in any **significant** way by his occupational exposure to coal dust. At one point, he indicated that the "primary" cause of Mr. Baird's death was the very severe damage to his coronary circulation; later he stated that pneumonia was the "direct" cause of death. Dr. Naeye acknowledged that Mr. Baird had very severe obstructive lung disease, which he felt was due to smoking, although he acknowledged that exposure to coal mine dust has a "minor" effect. He also stated that smoking damages the defense mechanisms in the lungs, increasing the likelihood of developing pneumonia, and making recovery much more difficult; but he also acknowledged that damage due to pneumoconiosis can have some effect on defense mechanisms in the lungs, albeit small compared with the effects of cigarette smoking. Clearly, although he repeatedly downplayed the role of pneumoconiosis in Mr. Baird's death, Dr. Naeye was not willing to categorically exclude it as a factor. Dr. Bush concluded that the autopsy slides showed only a moderate degree of pneumoconiosis, which he felt was not consistent with disabling disease. But he did not explain the basis for excluding this "moderate" degree of pneumoconiosis as a factor in Mr. Baird's respiratory impairment. And while he subscribed to the concept that pneumoconiosis did not cause, contribute to, or in any way hasten Mr. Baird's death, he also said that his pneumoconiosis had no "significant" effect on his course before death, as it was too limited in degree and extent to have made any "significant" contribution to his medical problems. The Act does not require that pneumoconiosis be the predominant or even significant factor resulting in death, only that pneumoconiosis play some part, however minor, in hastening that death. I find that the statements of Dr. Naeye and Dr. Bush are contradictory and equivocal, and not persuasive evidence that pneumoconiosis played absolutely no role in Mr. Baird's death.

[6] Unlike the Employer, the Claimant, through her representative, specifically designated five letters by Dr. Parrish from Mr. Baird's living miner's claim for consideration in this claim. As Dr. Parrish was Mr. Baird's treating physician, these records do not exceed the Claimant's evidentiary limitations. These letters clearly document and support Dr. Parrish's conclusion that Mr. Baird's severe obstructive impairment was due to his pneumoconiosis, as well as his history of cigarette smoking, and that his obstructive impairment was a factor in his respiratory death. Nevertheless, even if I were to exclude them from consideration, I would find that the reports from Dr. Parrish exclusive of those in the living miner's claim are more than sufficient, in other words, well-reasoned and supported, to support a finding that Mr. Baird's death was due to his pneumoconiosis, as required by the Act.

Complicated Pneumoconiosis

If the Claimant could establish that Mr. Baird suffered from complicated pneumoconiosis, she would be entitled to an irrebuttable presumption that Mr. Baird's death was due to pneumoconiosis. Cite. But while Dr. Parrish has repeatedly stated that Mr. Baird suffered from complicated pneumoconiosis, I find that the evidence of record is not sufficient to support such a finding.

Although the term does not appear in the statute, a miner can establish "complicated pneumoconiosis" if he suffers from a chronic dust disease of the lung which:

> (a) When diagnosed by chest x-ray . . . yields one or more large opacities (greater than 1 centimeter in diameter) and would be classified in Category A, B, or C…; ***or***
>
> (b) When diagnosed by biopsy or autopsy, yields massive lesions in the lung; ***or***
>
> (c) When diagnosed by means other than those specified in paragraphs (a) and (b) of this section, would be a condition which could reasonably be expected to yield the results described in paragraph (a) or (b) of this section had diagnosis been made as therein described: Provided, however, That any diagnosis made under this paragraph shall accord with acceptable medical procedures.

20 C.F.R. §718.304 (emphasis added); s*ee Eastern Associated Coal Corp. v. Director, OWCP*, 220 F.3d 250 (4th Cir. 2000). The Fourth Circuit has recently described the appropriate analysis under Section 21(c)(3) of the Act and the implementing regulations at 20 C.F.R. §718.304:[7]

> While 30 U.S.C. §921(c)(3) sets forth, in clauses (A), (B), and (C), three different ways to establish the existence of statutory complicated pneumoconiosis for purposes of invoking the irrebuttable presumption, these clauses are intended to describe a single, objective condition. . . And, because prong (A) sets out an entirely objective scientific standard—i.e. an opacity on an x-ray greater than one centimeter—x-ray evidence provides the benchmark for determining what under prong (B) is a massive lesion and what under prong (C) is an equivalent diagnostic result reached by other means.
>
> Prongs (A), (B), and (C) are stated in the disjunctive; therefore a finding of statutory complicated pneumoconiosis may

[7] It is important to note that Section 21(c)(3) of the Act, 30 U.S.C. §921(c)(3), and Section 718.304 of the implementing regulations, 20 C.F.R. §718.304, are virtually identical in language, and the Fourth Circuit has treated them as interchangeable for purposes of invoking the irrebuttable presumption. *See Eastern Associated Coal*, 220 F.3d 250.

> be based on evidence presented under a single prong. But the ALJ must in every case review the evidence under each prong of §921(c)(3) for which relevant evidence is presented to determine whether complicated pneumoconiosis is present. Evidence under one prong can diminish the probative force of evidence under another prong if the two forms of evidence conflict. Yet, a single piece of relevant evidence can support an ALJ's finding that the irrebuttable presumption was successfully invoked if that piece of evidence outweighs conflicting evidence in the record. Thus, even where some x-ray evidence indicates opacities that would satisfy the requirements of prong (A), if other x-ray evidence is available or if evidence is available that is relevant to an analysis under prong (B) or (C), then all of the evidence must be considered and evaluated to determine whether the evidence as a whole indicates a condition of such severity that it would produce opacities greater than one centimeter in diameter on an x-ray. Of course, if the x-ray evidence vividly displays opacities exceeding one centimeter, its probative force is not reduced because the evidence under some other prong is inconclusive or less vivid. Instead, the x-ray evidence can lose force only if other evidence affirmatively shows that the opacities are not there or are not what they seem to be, perhaps because of an intervening pathology, some technical problems with the equipment used, or incompetence of the reader.

Eastern Associated Coal, 220 F.3d at 255-6 (internal quotations and citations omitted). The Fourth Circuit emphasized that the parties should not assume "that the statutory definition of 'complicated pneumoconiosis' must be congruent with a medical or pathological definition." *Id.* at 257. Instead, it is important to remember in the determination of complicated pneumoconiosis that the presumption under 20 C.F.R. §718.304 "is triggered by a congressionally defined condition." *Id.* In other words, invocation of the irrebuttable presumption does not require any additional clinical finding if prong (A), (B), or (C) is met.

The Court noted that the statute creating the irrebuttable presumption of causation does not refer to the condition as "complicated pneumoconiosis," or to a medical condition that doctors have independently called complicated pneumoconiosis. As the Court stated.

> [T]he presumption under § 921(c)(3) is triggered by a congressionally defined condition, for which the statute gives no name but which, if found to be present, creates an irrebuttable presumption that disability or death was caused by pneumoconiosis. . . . In short, the statute betrays no intent to incorporate a purely medical definition.

Eastern Associated Coal Corporation, 250 F.3d at 257.

Thus, if the Claimant meets the congressionally defined condition, that is, if he establishes that he has a condition that manifests itself on x-rays with opacities greater than one centimeter, he is entitled to the irrebuttable presumption of total disability due to

pneumoconiosis, unless there is affirmative evidence under prong A, B, or C that persuasively establishes either that these opacities do not exist, or that they are the result of a disease process unrelated to his exposure to coal mine dust.

In his letter dated March 13, 2002, Dr. Parrish indicated that Mr. Baird's x-rays showed conglomerate lesions that he would rate as Category C under the ILO classifications. However, he did not refer to a specific x-ray interpretation, nor are there any ILO interpretations anywhere in the exhibit file that document Category C opacities.

In contrast, Dr. Naeye stated that, in order to show up as a mass of one centimeter on x-ray, a mass or conglomerate lesion must measure at least two centimeters on autopsy, because the x-ray passes through the edges of the mass. Because there were no such masses or lesions found on autopsy, Dr. Naeye did not make any findings of complicated pneumoconiosis. In other words, Dr. Naeye concluded that the masses identified by Dr. Harrison would not appear on x-ray as areas of conglomeration at least one centimeter in diameter.

No other physician has indicated that the masses and areas of conglomeration found by Dr. Harrison on autopsy would translate to opacities of at least one centimeter on x-ray. Nor has any physician addressed the question of whether Mr. Baird's autopsy findings translate into a finding of massive lesions that would result in opacities of at least one centimeter on x-ray.

I find that the Claimant has not established that Mr. Baird had a condition of such severity that it would have resulted in x-ray findings of opacities of at least one centimeter in diameter, which is the congressional definition of complicated pneumoconiosis under the Act. Therefore, the Claimant is not entitled to the irrebuttable presumption that Mr. Baird's death was due to pneumoconiosis.

CONCLUSION

Based on the foregoing, I find that the Claimant has established by a preponderance of the medical evidence that Mr. Baird suffered from coalworkers' pneumoconiosis that arose from his coal mine employment, and that his death was due to pneumoconiosis. Thus, the Claimant, as his survivor, is entitled to benefits under the Act.

ORDER

Based on the foregoing, IT IS HEREBY ORDERED that the claim of Martina D. Baird for benefits under the Black Lung Benefits Act is granted.

IT IS FURTHER ORDERED that the Employer, Key Mining Inc., and its insurer, American Mining Insurance Co., shall pay to the Claimant all benefits to which she is entitled under the Act.

SO ORDERED.

A

LINDA S. CHAPMAN
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: Pursuant to 20 C.F.R. 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 (thirty) days from the date of this decision by filing a Notice of Appeal with the Benefits Review Board at P.O. Box 37601,Washington, D.C. 20013-7601. *A copy of a Notice of Appeal must also be served on the Associate Solicitor for Black Lung Benefits, 200 Constitution Avenue, N.W., Room N-2117, Washington, D.C. 20210.*